Non-specific low back pain

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goals of individuals and communities to achieve optimum quality and quantity of life. There is increasing evidence that such a health system should be based on strong primary health care, that uses an effective community-oriented primary care model,[4] addressing ways to reduce the causes of NCDs and tackling social and political issues at the local, national, and international level because of its emphasis on community input.

Earlier this year, Richard Horton[5] commented that "There has been an argument for several decades now to drop vertical disease programmes…and replace them with schemes that emphasise health systems strengthening… Health systems approaches to aid may be intellectually correct, but they are politically problematic." A comprehensive integrated strategy based on primary health care to tackle NCDs creates a unique opportunity to make the switch.

We declare that we have no conflicts of interest.

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Non-specific low back pain

In their Seminar on low back pain, Federico Balagué and colleagues (Feb 4, p 482)[1] conclude that (occupational) mechanical factors are unlikely to be independently causative of low back pain. This far-reaching conclusion is based on reviews of published epidemiological studies and on the relation between evidence of tissue injury on imaging and low back pain.

In terms of epidemiology, Balagué and colleagues base their conclusion on a series of reviews by Wai, Roffey, Bishop, Kwon, and Dagenais. These reviews have been criticised for several reasons.[2,3] First, they rely on application of the Bradford-Hill criteria to single epidemiological studies, whereas these criteria were proposed to help assess the evidence for causality across studies from different disciplines. Second, other reviews[4] have reached contrasting conclusions. Third, in the studies on which the reviews were based, exposure to mechanical loading was incomplete—ie, not encompassing intensity, frequency, and duration—and was based on inaccurate proxy measures. Where exposure has been better characterised, strong relations are seen.[5]

Balagué and colleagues furthermore use the lack of a one-to-one relation between back pain and structural damage to the spine as an argument against the relevance of mechanical injury in the origin of low back pain. Such an argument could be used similarly to deny the relation between smoking and lung cancer.

Neglect of occupational, mechanical loading as a causal factor in low back pain is not based on evidence and might seriously hamper effective prevention and management.

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Authors' reply

Before we respond to the issues raised by J H van Dieën and colleagues, we would like to point out a couple of minor inaccuracies in their letter. Our Seminar was not about "low back pain" (all-cause) but about non-specific low back pain, as defined in the opening paragraph. This is not just a semantic issue. There is an important distinction between the two, and one that is highly relevant in this context. The biological plausibility of a mechanical role in (some) back pain—on the basis of experimental or laboratory studies including those by van Dieën and colleagues—mainly concerns specific types of low back *injury* such as acute prolapsed disc, fracture, etc. The second inaccuracy is that our conclusion (p 488) makes no reference to any specific causative factors in back pain; it acknowledges the effect of physical and environmental factors, among others.

We have read the earlier letters by van Dieën, Kuijer, and others criticising the Dagenais group's systematic reviews and we refer the interested reader to the eloquent

rebuttals provided by the authors of those reviews.

Our colleagues might not be familiar with the specifications given to authors in writing for *The Lancet*'s Seminar series. The remit is to create an article that is "clinically focused and up-to-date", with a limit on the number of words and the quantity, nature, and age of the references cited. Owing to their uppermost position in the hierarchy of evidence, systematic reviews are relied on heavily. These prerequisites preclude detailed discussion of the strengths and weaknesses of the cited works. Our aim was to fulfil our remit while stimulating reflection and further enquiry by the interested reader.

In the Seminar, we highlight the fact that epidemiological studies do not seem to support the notion of mechanical factors being independently causative of low back pain. Despite the enormous amount of research done in the specialty of biomechanics and ergonomics, there has been no notable improvement in the burden of non-specific low back pain. As clinicians, we are acutely aware of the potentially detrimental side-effects of repeated messages about "ergonomically correct behaviour" that in some patients merely serve to promote kinesiophobia or fear avoidance behaviour.

With respect, we think the analogy with smoking and lung cancer is rather trite; one need only to look at another important Bradford-Hill aspect of causality, "experiment (reversibility)", to realise that the benefits of ergonomic prevention programmes for back pain[1] are in no way comparable to those of smoking cessation for cancer.[2,3]

Concerning the final paragraph of van Dieën and colleagues' letter, we deny any suggestion that occupational, mechanical loading should be neglected within the context of low back pain; however, for the aforementioned reasons, together with the finding that a high proportion of teenagers report non-specific low back pain (yet have zero exposure to occupational loading) and data apportioning the contribution of suspected explanatory variables (genetic, mechanical, other),[4,5] we maintain that a major causal role for occupational, mechanical loading remains questionable.

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Intravenous salbutamol in ARDS and increased mortality

In the BALTI-2 trial, Fang Gao Smith and colleagues (Jan 21, p 229)[1] report an increase in 28-day mortality after a 7-day infusion of salbutamol compared with placebo in patients with early acute respiratory distress syndrome (ARDS). Major discrepancies between predicted and observed mortality make interpretation of this trial difficult, especially when these figures are compared with those reported in Smith and colleagues' previous study (BALTI),[2] which was used to calculate the sample size.

The observed mortality rates (23% in the placebo group and 34% in the intervention group) are lower than those predicted by the severity of illness (assessed by the Acute Physiology and Chronic Health Evaluation [APACHE II]), resulting in very low standardised mortality ratios (0·54 in the placebo group and 0·79 in the treated group). The observed mortality rates are also different from the figures reported in the BALTI study: 66% and 58% in the placebo and treated groups, respectively. These data raise a logical question as to the true severity of disease in these patients, and whether these findings should be extrapolated to real life. Indeed, although BALTI-2 included patients with the most severe disease (only ARDS, whereas those with either acute lung injury or ARDS were included in BALTI), Smith and colleagues reported similar PaO_2/FiO_2 ratios, lower APACHE II score, and overall a very low 28-day mortality.[1]

Moreover, careful examination of the survival curve shows that there was a non-significant increase in mortality during the drug infusion period (up to 7 days): 9·1% and 9·2% mortality by day 6 in the treated group and in the placebo group, respectively. This finding adds to the difficulties in interpreting the results of this study.

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